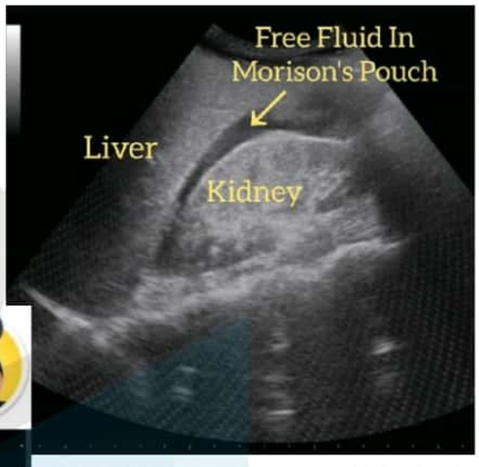
Abdominal Trauma

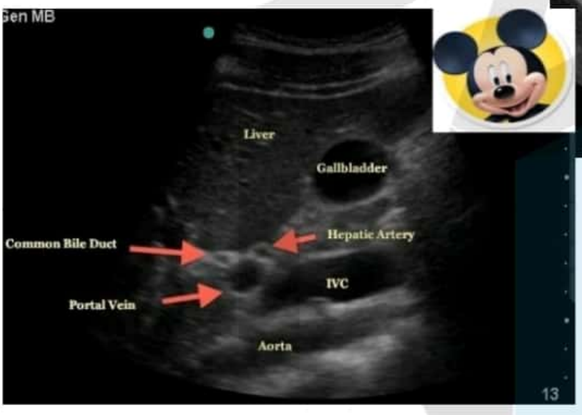
Case: 45 year man meets with a RTA and is rushed to the ER with suspected abdominal injury. Patient is hypotensive and FAST is positive. On laparotomy, liver appears lacerated and is actively bleeding. Surgeon performs pringles, plugging and packing but after 20- 30 mins notices blood oozing from the posterior surface. Which is the most appropriate next step?

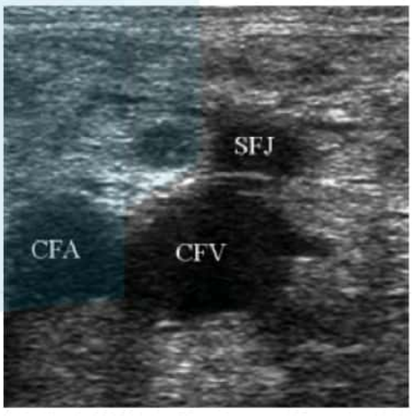
This USG done in Right flank shows presence of fluid in the Morrison's pouch.

This is called as FAST +ve



Portal Triad USG→ Mickey mouse sign or Exclamation Dot sign





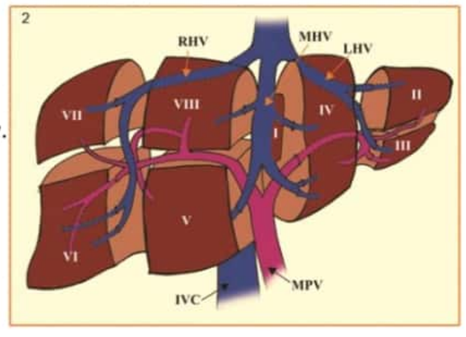
Mickey Mouse sign is also seen in-

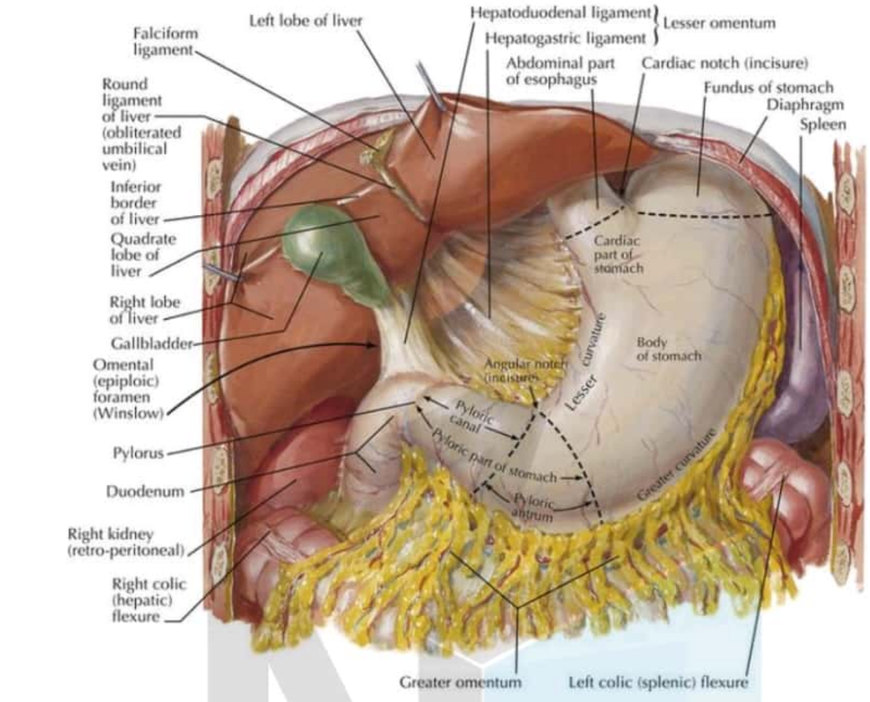
USG done during Deep Vein Thrombosis

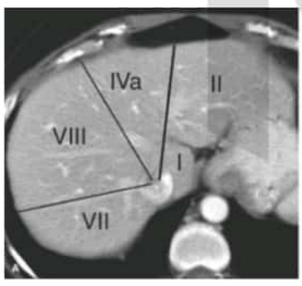
ANATOMY OF LIVER

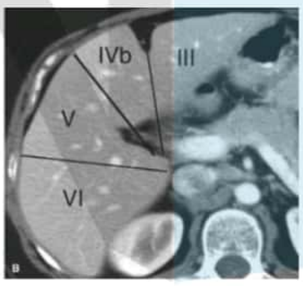
- Couniad's Classification
  - Based on Portal vein and Hepatic artery.
  - Caudate Lobe: Couniad Lobe 1
  - Quadrate Lobe: Couniad Lobe 4B
- Between the caudate and the quadrate lobes is present the area- Porta Hepatis.

1

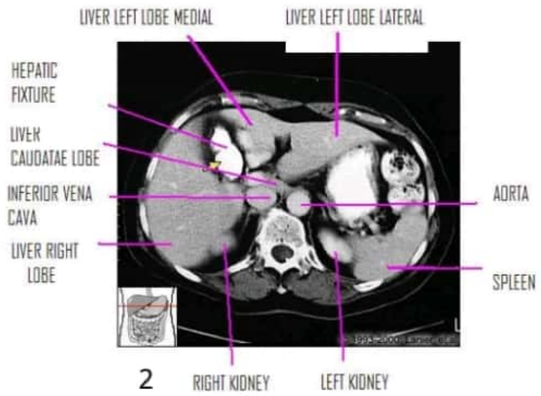




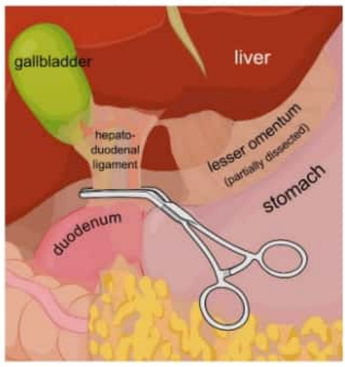




Caudate Lobe hypertrophy is always associated with BUDD-CHIARI SYNDROME

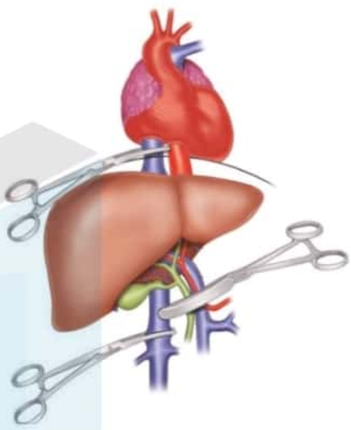


Coming back to the case...



The **Pringle maneuver** is a surgical maneuver used in some abdominal operations. A large atraumatic hemostat is used to clamp the **hepatoduodenal ligament** (free border of the lesser omentum) interrupting the flow of blood through the **hepatic artery, the portal vein & Bile duct** and thus helping to control bleeding from the liver. More commonly, in the absence of soft clamp, manual compression of the hepatoduodenal ligament is performed from the **Foramen of Winslow.**

If bleeding does not stop with the Pringle's Maneuver, the next best step to do is...



HEANEY's MANEUVER: The Heaney maneuver is performed by clamping both the suprahepatic and infrahepatic inferior vena cava while simultaneously applying the Pringle maneuver. During the Heaney maneuver, clamping of the inferior vena cava can lead to cardiac arrest because of the sudden decrease in cardiac preload.

Other Maneuvers:

1. Mattox Maneuver: mobilization of the descending colon to the midline to expose the abdominal aorta.

2. Cattle Maneuver: mobilization of the ascending colon to the midline.

3. Kocher maneuver: dissection of the lateral peritoneal attachments of the duodenum to allow inspection of the duodenum, pancreas, and other retroperitoneal structures over to the great vessels.

Brisbane Terminology of Liver Anatomy and Resections

Anatomic Term	Couinaud Segments	Surgical Resection
Right hemiliver/ right liver	5-8	Right hepatectomy
Left hemiliver/left liver	2-4	Left hepatectomy
Right anterior section	5, 8	Right anterior sectionectomy
Right posterior section	6, 7	Right posterior sectionectomy
Left medial section	4	Left medial sectionectomy *or* Resection of segment 4
Left lateral section	2, 3	Left lateral sectionectomy *or* Bisectionectomy 2, 3
	4, 5, 6, 7, 8	Right trisectionectomy *or* Extended right hepatectomy
	2, 3, 4, 5, 8	Left trisectionectomy *or* Extended left hepatectomy

Infectious Diseases of liver

Case: A 50-year-old woman complains of a 4-month history of right-sided abdominal pain and nausea. Her vital signs are stable, and she is afebrile. Her physical examination is unremarkable except for hepatomegaly. Ultrasound of the abdomen shows an 8-cm wellcircumscribed cyst with a rosette appearance. What is the preferred treatment of this patient?

CLINICAL FEATURES	PYOGENIC ABSCESS	AMEBIC ABSCESS	HYDATID CYST
Age	> 50 years	20-40 years	40 – 50 years
Male-to-female ratio	3:2	10: 1	1: 1
Solitary versus multiple	50% solitary 50% multiple	80% solitary	Solitary
Location	Right hemi liver	Right hemi liver	Right hemi liver
Travel in endemic area	No	Yes	Yes
Diabetes association	Yes (more common)	Uncommon	Uncommon
Alcohol use	Common	Common	Uncommon
Jaundice	Common	Uncommon	Uncommon (8%)
Elevated bilirubin	Common	Uncommon	Uncommon
Elevated ALP	Common	Common	Uncommon
Treatment of choice	Image guided aspiration + antibiotics 10-14 days, upto 6 weeks	High dose Metronidazole (750 mg TID)	PAIR + pericystectomy with omentopexy

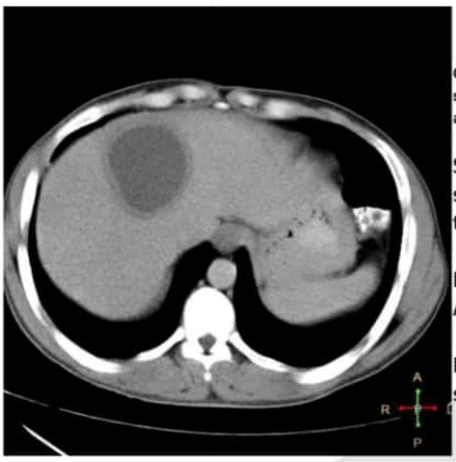
CLUSTER SIGN

Heterogenous lesion with multiple central non-enhancing pus pockets forming a cluster

Dx: PYOGENIC liver abscess.

Also seen in this CT is:

The **double target sign** is a characteristic imaging feature of liver abscess on contrast enhanced CT scans, in which a central, fluid-filled low attenuation lesion is surrounded by a high attenuation inner rim and a low attenuation outer ring.

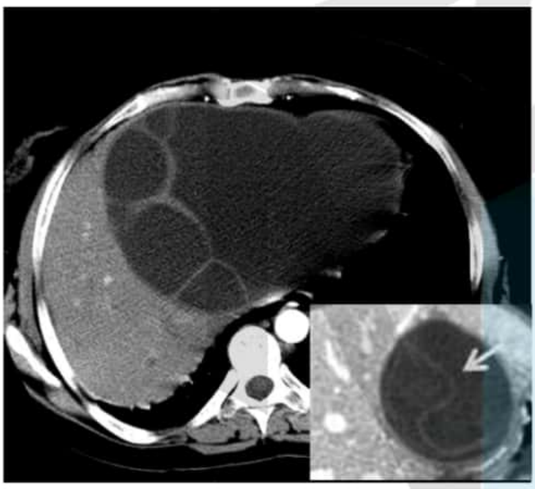


Double target sign:
Central Dark shadow surrounded by a hyper-attenuated rim.

Solitary cystic lesion surrounded by soft tissue edema

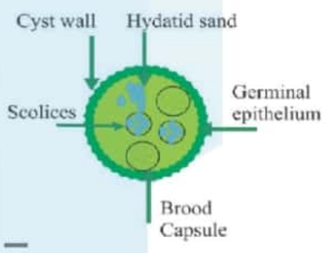
Dx: Amebic Liver Abscess.

Double Target is also seen in pyogenic



Free Floating membranes in the cyst

Dx: Hyatid Cyst

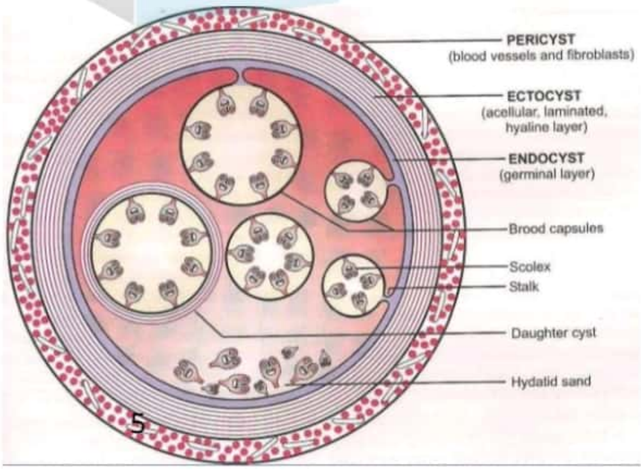


PERICYST is derived from the host.

ENDOCYST and ECTOCYST is derived from the parasite.

In PAIR therapy, you puncture the PERICYST, aspirate the material, scolicidal agent is introduced to prevent seeding during Pericystectomy.

We don't breech the Ectocyst or Endocyst.



Benign LIVER TUMORS

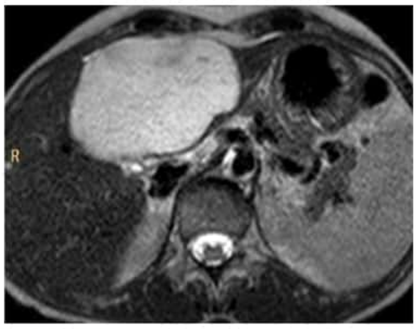
Case: A 28-year-old asymptomatic white woman is incidentally found to have a 3.5- cm hypervascular lesion with a central scar in the right lobe of her liver. On delayed images, there is increased uptake of contrast material in the scar in comparison with the surrounding liver parenchyma. She is otherwise healthy and takes no medications. Liver enzyme and α-fetoprotein levels are within normal limits. Which of the following is the most appropriate management of this patient?

Ans- Observation

	Hemangioma	FNH	Hepatic Adenoma
Prevalence	20%	1%	0.05%
Pathogenesis	Vascular malformation	Hyperplasia response to vascular malformation	OCP, non-alcoholic steatohepatitis, **type I & III glycogen storage disease**
AFP	normal	Normal	Normal
complications	Mass effect, Kasbach Merritt syndrome (if >5cm)	Mass effect, Torsion if pedunculated	Mass effect, bleeding, shock
Risk of malignant transformation	No	No	Yes (size> 5cm)
Radiological feature	Light bulb	Central stellate scar	Distorted architecture
Treatment	Enucleation (symptomatic)	Observation (resection in symptomatic and pedunculated FNH)	Excision

- M/C Malignancy in Liver: Metastasis from other cancers
- M/C Primary Malignancy in Liver: Hepatocellular Cancer
- M/C Primary Malignancy in Liver in Children: Hepatoblastoma
- M/C Benign Liver Tumour: Hemangioma
- Malignant Condition of liver where AFP is normal: FIBROLAMELLAR variant of HCC
- Lesions where Enucleation is the treatment of choice: (HEMA in LIC)
  - Hemangioma of liver
  - Leiomyoma of pancreas
  - Insulinoma- at the head of pancreas
  - Chylo-lymphatic cyst

Radiological Findings

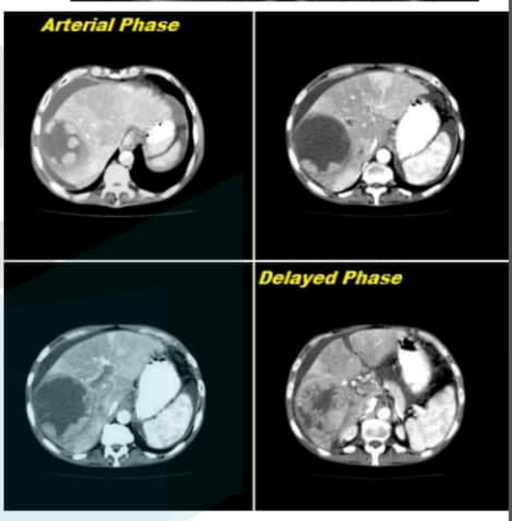


1. Hemangioma of Liver

IOC- MRI → Hyperintense bright signal on T2 weighted MRI, called as LIGHTBULB appearance of Hemangioma of liver.
Though, Hepatic Hemangioma is an incidental finding.

[LIGHTBULB is also seen in PHEOCHROMOCYTOMAs]

Triple Phase CT → Arterial phase: Peripheral Nodular enhancement. In Delayed phase it shows Centripetal Filling



2. Focal Nodular Hyperplasia [FNH]

Enhancement in the arterial phase but- Central Scar shows delayed enhancement on Triple phase CT

FNH is rich in Kupffer cells, henceforth in Tc-Colloid Scan it shows HOT lesion.

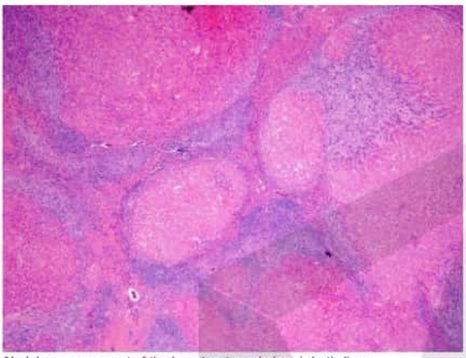
Lesions where you see a 'Central Stellate Scar'

1. Focal Nodular Hyperplasia
2. Fibrolamellar HCC
3. Serous cystadenoma of pancreases
4. Renal Oncocytoma

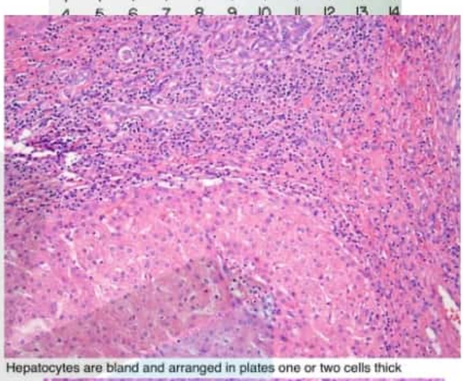
Pathological Findings

1. Focal Nodular Hyperplasia

Gross: Resected specimen showing lobulated contours and a central stellate scar

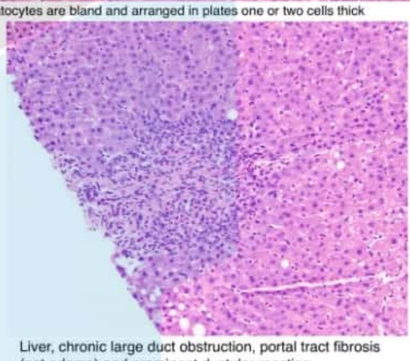


Nodular arrangement of the hepatocytes mimics cirrhotic liver



Hepatocytes are bland and arranged in plates one or two cells thick

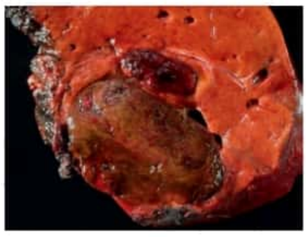
The high-power view also shows – Proliferation of glands in the septal regions known as the 'DUCTULAR REACTION'

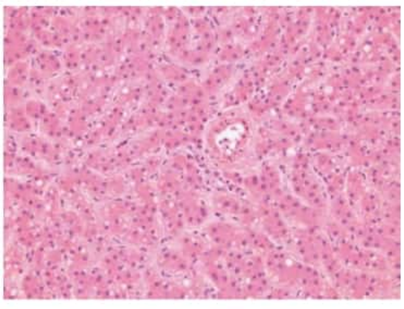


Liver, chronic large duct obstruction, portal tract fibrosis (not edema) and prominent ductular reaction.

2. Hepatocellular Adenoma

Gross: Resected specimen showing a well-define tan mass in the liver. No Scarring is present.



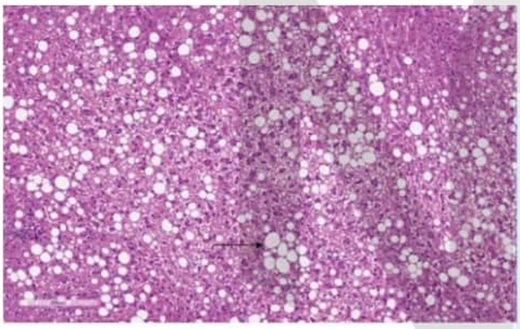


Microscopy: thin cords of hepatocytes, with an arterial vascular supply *and* no portal tracts.

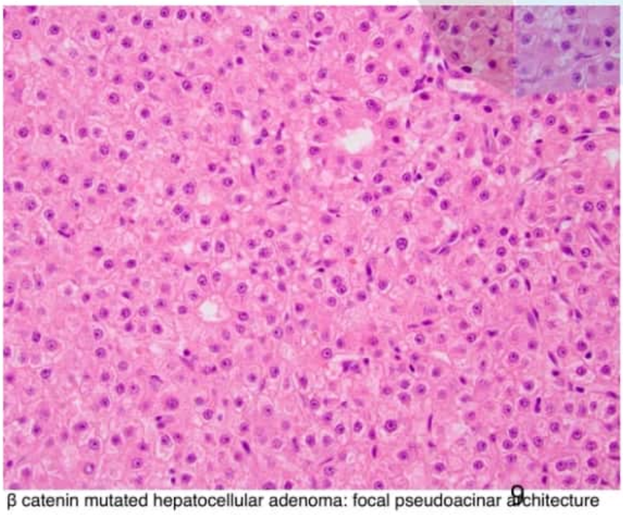
Types of Hepatic Adenoma-

1. HNF- 1α mutated HA-H (No risk of malignancy) = fatty change -----MOST COMMON TYPE
2. B-catenin mutated, HA-B = pseudo-acinus formation (Inc risk of malignancy)
3. Inflammation HA-I
4. Sonic hedge-hog HA (HA-Sh)
5. HA U (Not Otherwise Specified)

Subtype		Frequency	Molecular findings
HA-H (HA-s)	HNF1α mutated hepatocellular adenoma	35%	Somatic mutations of TCF1(HNF1A) gene, heterozygous germline mutations (<5%) of CYP1B1 gene
HA-B	β-catenin mutated hepatocellular adenoma	10%	β-catenin gene activating mutations (Exon 3 vs Exon 7-8 mutations)
HA-I	Inflammatory hepatocellular adenoma	35%	Gain of function mutations of the IL6ST gene; 10% with co-existing β-catenin gene mutations
HA-sh	Sonic hedgehog hepatocellular adenoma	5%	Activation of Sonic Hedgehog pathway via fusion of promoter of INHBE with GLI1
HA-U	Hepatocellular adenoma, not otherwise specified	<7%	No specific mutations



Fatty change in Hepatic Adenoma HNF1 Alpha mutated.



β catenin mutated hepatocellular adenoma: focal pseudoacinar architecture

Differentiated from the malignant tumors showing pseudoacinus by the absence of nuclear atypia.

Liver Malignancy

Case: A 55-year-old woman with a history of colorectal cancer 10 years ago, treated with right hemicolectomy and chemotherapy and with no evidence of disease since then, presents with vague abdominal pain. Temperature is 98.7°F, pulse 67 beats/ min, blood pressure 132/78 mmHg, and saturation 99% on room air. Labs are remarkable for AST 67 IU/L, ALT 83 IU/L, and carcinoembryonic antigen (CEA)-27 ng/mL. Triple-phase contrast CT shows a new peripheral liver mass that slightly enhances on arterial phase. She has hypertension that is well controlled with medications. What are the most likely diagnosis and most appropriate management of her liver lesion?

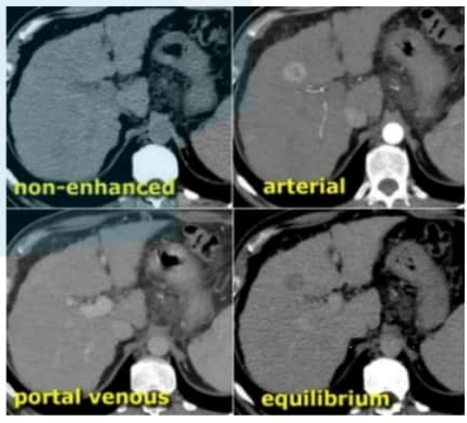
Ans- Metastatic Colorectal cancer, resection

POINTS IN FAVOUR OF COLORECTAL METASTASIS

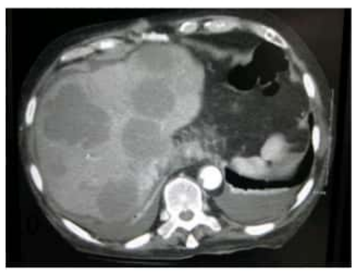
- history of primary cancer
- peripheral residing lesions
- multiple lesions
- hypovascularity (hence only slightly enhancing compared with adjacent liver parenchyma)
- CEA is usually elevated as well if colorectal cancer is the primary

Radiological Findings

- Triple-phase CT (includes an arterial phase, a portal venous phase, and a late equilibrium phase)
- Normal liver has a dual blood supply- Portal Vein (Predom) and Hepatic Artery
- Malignancies have a predominant blood supply from Hepatic Artery
  - HCC is HYPERvascular
  - Metastasis is HYPOvascular



HCC: Arterial phase- Enhancement
Venous phase- Washout
Equilibrium phase- Capsule can be seen



Secondary Metastasis in liver
(M/C from Colorectal Carcinoma)
Finding- HYPOVASCULAR
Hypodense in Portal Venous phase
(Except- if Carcinoid Tumor- Hypervascular)

Resection criteria

Minimal FLR volume required

20% in normal liver

30% in damaged liver

40% in cirrhotic liver

Margin of resection in liver : 10mm (1cm)

FLR: Future Liver Remanent (after the resection)

Milan's Criteria: for Liver TRANSPLANTATION

Milan Criteria (Mazzaferro et al, 1996)

- Single tumor ≤ 5 cm, or
- 2-3 tumors none exceeding 3 cm, and
- No vascular invasion and/or extrahepatic spread

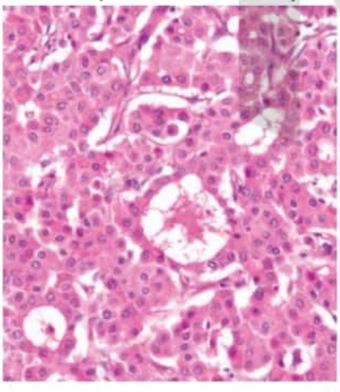
Pathological Findings of HCC

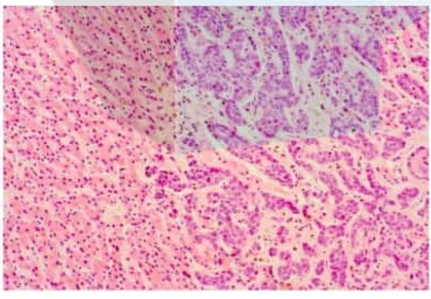
Gross: Liver removed at autopsy showing a unifocal neoplasm replacing most of the right hepatic lobe.

The tumour may have intrahepatic spread- showing as satellite lesions in the liver.

Tumours appear greenish-yellow due to the bile.

Microscopy: Malignant hepatocytes growing in distorted versions of normal architecture, including large pseudoacinar spaces (malformed, dilated bile canaliculi) and thickened hepatocyte trabeculae.





HCC

- Genetics
  - P53 : Most common mutation
  - Beta catenin (2nd MC)
- Precursor Lesions
  - Increased risk of malignancy: Hepatic adenoma, Small cell Change, High grade dysplastic nodule, Large cell change (If occurs in HBV)
  - Uncertain: Low grade Dysplasia

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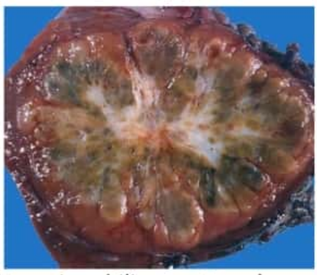
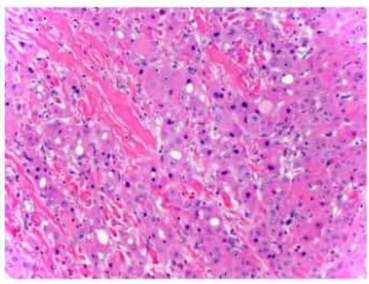
The malignant cells of this hepatocellular carcinoma (seen on the right) are well differentiated and interdigitate with normal hepatocytes arranged in regular cords (seen at the left).

Fibrolamellar HCC





Gross: Central Stellate Scar

Microscopy: Acellular Collagen due to scarring with atypical hepatocytes (prominent nucleoli, nuclear atypia)

Also, presence of PALE bodies (light eosinophilic structures) containing Fibrin (Non-specific finding)

HCC
Pushing type

HCC
Hanging type

HCC
diffuse
infiltrating type

Case: A 69-year-old woman with Child's C cirrhosis has an elevated AFP and a new liver mass on ultrasound. Triplephase contrast CT confirms a 3.5 ×4-cm2 mass with arterial enhancement and venous washout. She is otherwise healthy with no cardiopulmonary disease. What is the next most appropriate step in workup and treatment?

A. Interventional radiology (IR) biopsy and pathology
B. Neoadjuvant chemotherapy
C. Liver resection
D. Liver transplant

Answer should be: supportive care

Child-Pugh Classification

FACTOR	NO. OF POINTS 1	2	3
Bilirubin (mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time (increased seconds)	1-3	4-6	>6
Ascites	None	Slight	Moderate
Encephalopathy	None	Minimal	Advanced

Class A, 5-6 points; class B, 7-9 points; class C, 10-15 points.

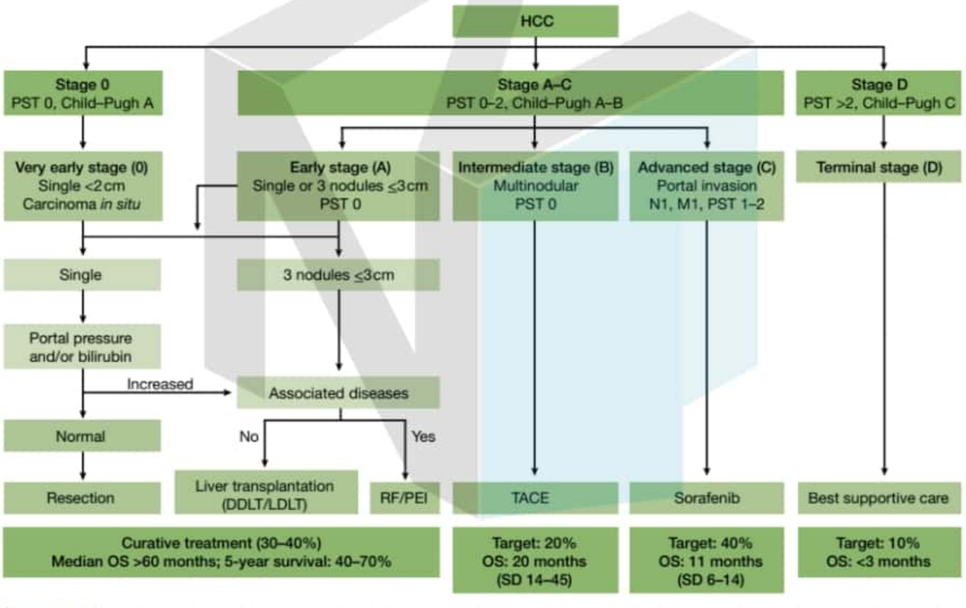
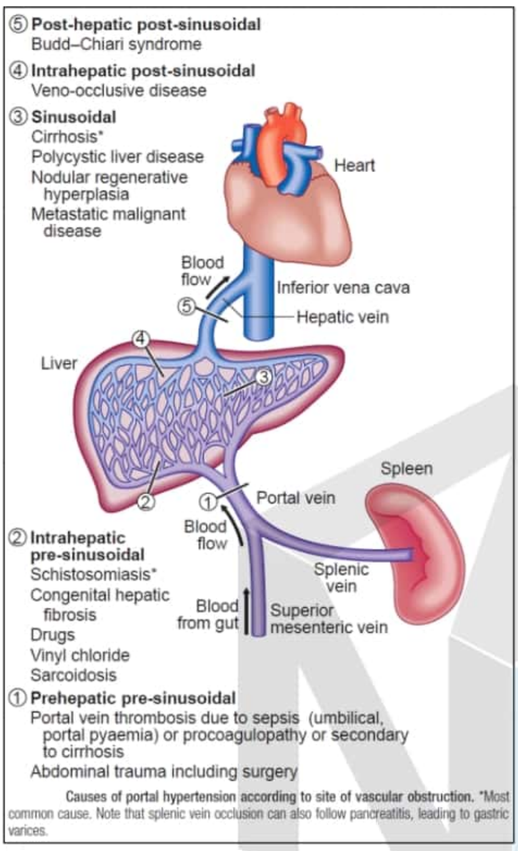


Figure 65.24 The Barcelona Clinic Liver Group staging system for the management of hepatocellular carcinoma (HCC). Patients with asymptomatic early tumours (stage 0–A) are candidates for curative therapies (resection, transplantation or local ablation). Asymptomatic patients with multinodular HCC (stage B) are suitable for chemoembolisation (TACE), whereas patients with advanced symptomatic tumours and/or an invasive tumoural pattern (stage C) are candidates for sorafenib. End-stage disease (stage D) includes patients with grim prognosis who should be treated by best supportive care. DDLT, deceased donor liver transplantation; LDLT, living donor liver transplantation; OS, overall survival; PEI, percutaneous ethanol injection; PST, ECOG performance status; RF, radiofrequency ablation; SD, standard deviation; TACE, transcatheter arterial chemoembolisation. (Reproduced with permission from Villanueva A. Medical therapies for hepatocellular carcinoma: a critical view of the evidence. *Nature Rev Gastroenterolol Hepatol* 2013; **10**: 34–42).

PORTAL HYPERTENSION

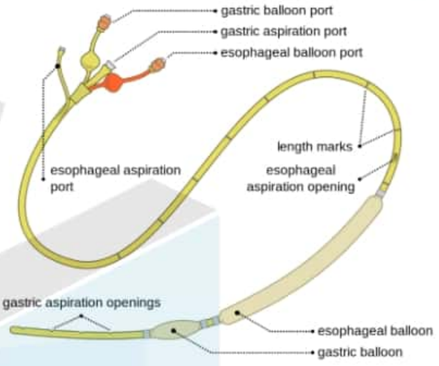
- Normal portal pressure : 5-8 mmHg
- Portal hypertension > 10mmHg
- Bleeding from oesophageal varices > 12mmHg
- Refractory variceal bleeding : > 3 failed attempts of EVL (Endovascular Variceal Ligation)



Causes of Portal Hypertension

Management of bleeding oesophageal varices

- Blood transfusion
- Correct coagulopathy
- Oesophageal balloon tamponade (Sengstaken–Blakemore tube)
- Drug therapy (terlipressin)
- Endoscopic sclerotherapy or banding
- Assess portal vein patency (Doppler ultrasound or CT)
- Transjugular intrahepatic portosystemic stent shunts (TIPSS)
- Surgery
  - Portosystemic shunts
  - Splenectomy and gastric devascularisation



Sengstaken-Blakemore tube

Esophageal Balloon: Inflated till 40mm Hg pressure
Gastric Balloon: Inflated with at least 300 mL of AIR

Deflate every 12 hours to avoid pressure necrosis

CHOLELITHIASIS

Question: Laparoscopic Cholecystectomy Is most strongly contraindicated in which of the following conditions:

a. Pregnancy
b. Previous upper abdominal surgery
c. Known CBD stones
d. **Recent MI with severe CAD notes on cardiac catheterization requiring full anti-coagulation**

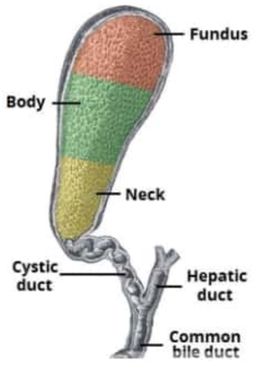
Ans: Minimum 6 months duration is required after an Myocardial infarction to take up the patient in any type of surgery

Gall Bladder Anatomy

Length: 7-10 cms, 3 cm wide

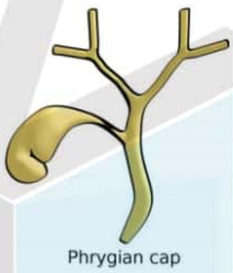
The gallbladder has a storage capacity of 30-50ml and, in life, lies anterior to the first part of the duodenum. It is typically divided into three parts:

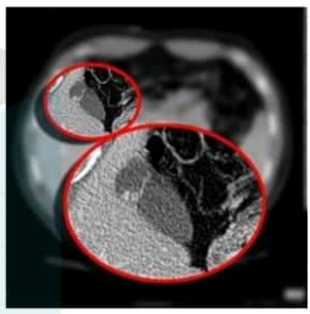
- **Fundus** – the rounded, distal portion of the gallbladder. It projects into the inferior surface of the liver in the mid-clavicular line.
- **Body** – the largest part of the gallbladder. It lies adjacent to the posteroinferior aspect of the liver, transverse colon and superior part of the duodenum.
- **Neck** – the gallbladder tapers to become continuous with the cystic duct, leading into the biliary tree.
  - The neck contains a mucosal fold, known as Hartmann's Pouch. This is a common location for gallstones to become lodged, causing cholestasis.



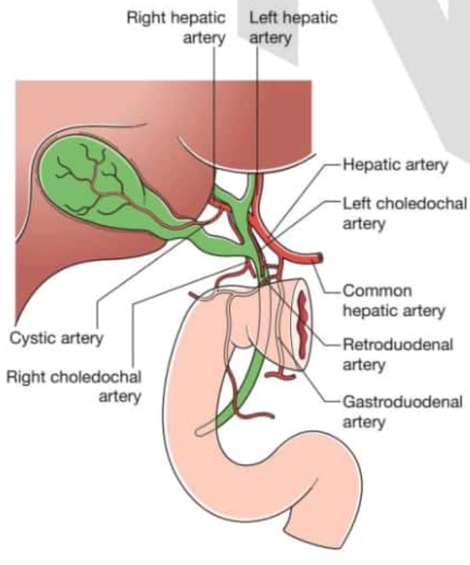
Congenital Anomalies:

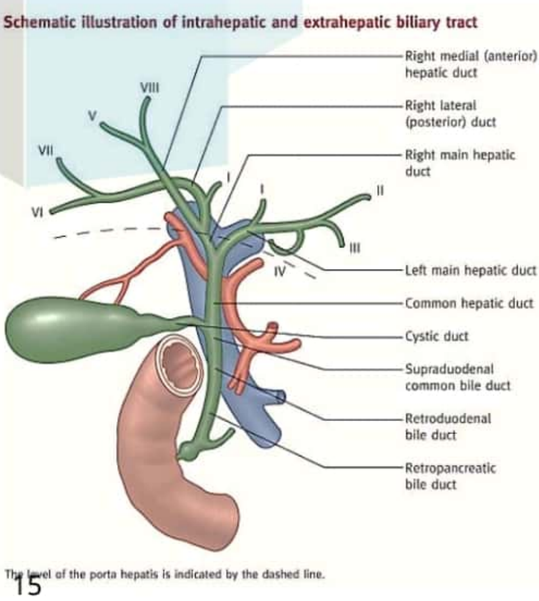
1. Agenesis
2. Septate GB
3. Double GB
4. Cysto-hepatic communication- ducts passing through bed of GB connected to intrahepatic bile ductules.
5. Phrygian cap- deformity of the fundus
6. Intrahepatic GB





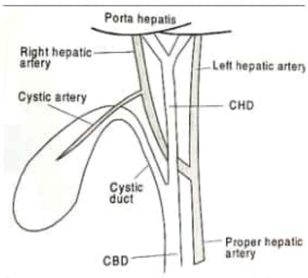
Biliary Tree Anatomy



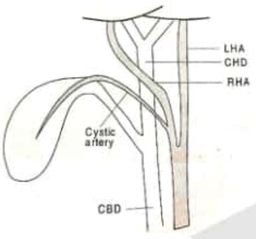


Anomalies

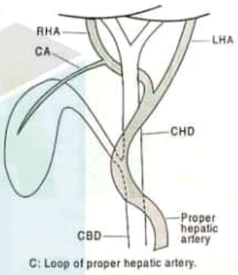
1. Right hepatic artery runs parallel to cystic duct & is in danger while ligating the duct.



A: Right hepatic artery running parallel to cystic duct.

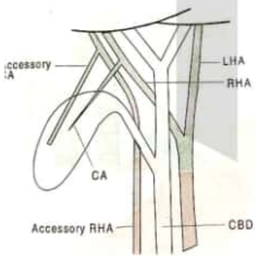


2. Cystic artery crosses in front of CHD, & this may be damaged while ligating the cystic artery.



C: Loop of proper hepatic artery.

3. Hepatic artery proper making a loop or caterpillar turn in front of cystic duct.



4. An accessory cystic artery arises from accessory right hepatic artery which arises from SMA.

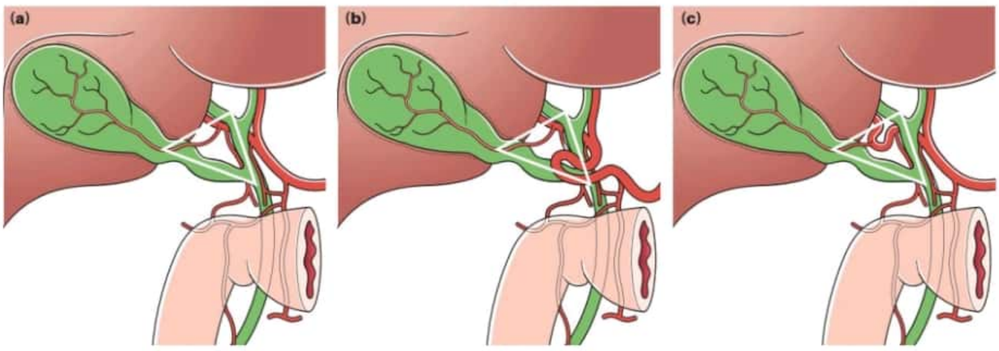
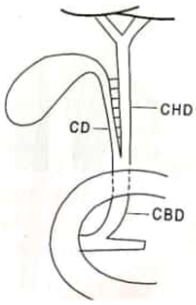


Figure 67.2 (a) Usual anatomy of Calot's triangle; (b) tortuous common hepatic artery; (c) tortuous right hepatic artery with short cystic artery; (b) and (c) are examples of so-called 'caterpillar turn' or 'Moynihan's hump', which if not recognised can lead to inadvertent arterial injury or bleeding during cholecystectomy.

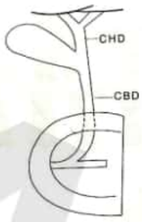
Variations in Duct System

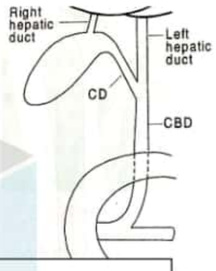
1. Cystic duct & CHD lie parallel to each other & are joined by connective tissue.
CHD may be damaged while ligating the cystic duct in cholecystectomy



2. Both cystic duct & CHD pass behind the duodenum to join behind the pancreas to form a short CBD.

3. Absent cystic duct – Neck of GB joins CHD.

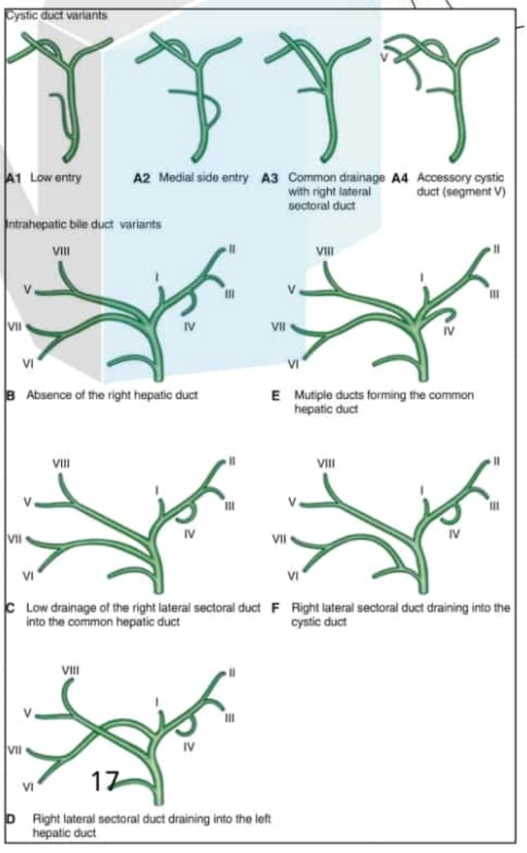




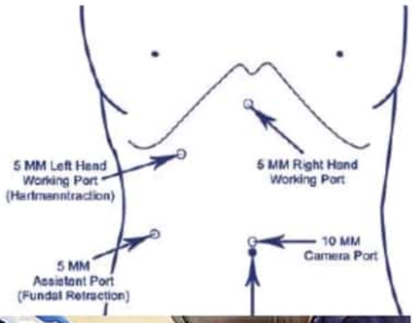
4. Right hepatic duct join GB. The left hepatic duct & cystic duct join to form CBD

Variations in the confluence of the left and right hepatic ducts.

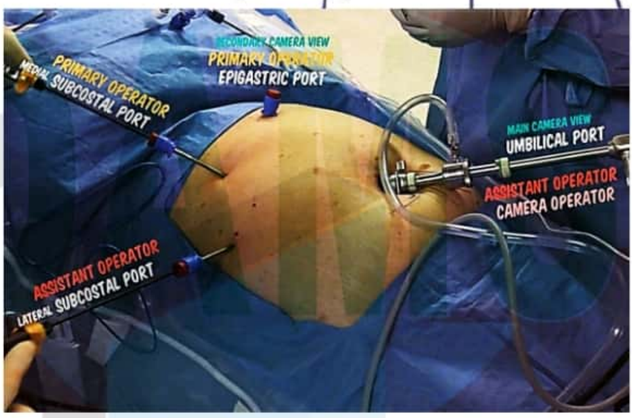
- **A,** Typical anatomy of the confluence.
- **B,** Trifurcation of left, right anterior, and right posterior hepatic ducts.
- **C,** Aberrant drainage of a right anterior (C1) or posterior (C2) sectoral hepatic duct into the common hepatic duct.
- **D-F,** Less common variations in hepatic ductal anatomy.


Cystic duct variants
A1 Low entry
A2 Medial side entry
A3 Common drainage with right lateral sectoral duct
A4 Accessory cystic duct (segment V)
Intrahepatic bile duct variants
B Absence of the right hepatic duct
E Mutiple ducts forming the common hepatic duct
C Low drainage of the right lateral sectoral duct into the common hepatic duct
F Right lateral sectoral duct draining into the cystic duct
D Right lateral sectoral duct draining into the left hepatic duct
17

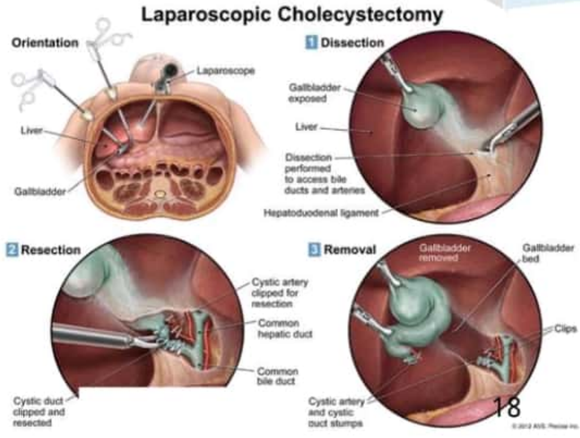
Laparoscopic Cholecystectomy


5 MM Left Hand Working Port (Hartmanntraction)
5 MM Right Hand Working Port
10 MM Camera Port
5 MM Assistant Port (Fundal Retraction)

- The standard technique of performing LC is to use 4 ports. The pneumoperitoneum is achieved by either closed Veress needle technique or open technique using a blunt trocar or a Hasson's trocar.
- A 10 mm telescope usually a 30 degree is used at the umbilicus either infra, intra or supraumbilical depending on patient's habitus and surgeon's preference.
- Another 10 mm trocar is used in the epigastrium which is the main right working port for the surgeon.
- One 5 mm trocar in the right lumbar region is used for gallbladder fundus traction and another 5 mm trocar in the right hypochondrium is used as left hand working port for the surgeon.

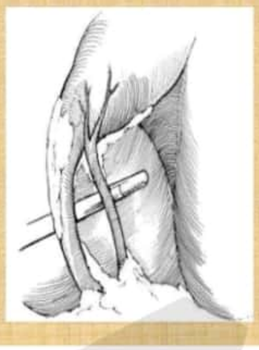


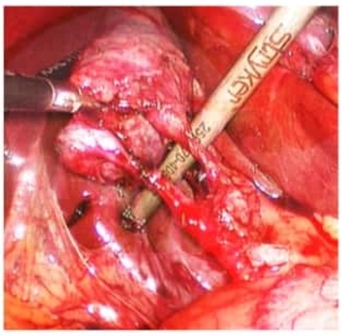
- With left hand Hartmann's pouch is retracted and with right hand anterior and posterior dissection is done in Calot's triangle and wide window is created.
- Critical view of safety is of utmost importance to prevent bile duct injury. Clipping of the cystic duct and cystic artery is achieved from 10 mm epigastric port. Gallbladder extraction is generally done from either epigastric or umbilical port.
- This technique can be named as "*10-10-5-5*" or "*4 ports standard LC*"



CRITICAL VIEW OF SAFETY

- Calot's triangle dissected free of all tissue except cystic duct & artery
- Base of liver bed exposed
- When this view is achieved, the two structures entering GB can only be cystic duct & artery
- Not necessary to see CBD





The plane of dissection of Calot's triangle is SUBSEROSAL in which the subserosal (SS) layer is divided into the inner layer of the SS (SS-Inner) layer consisting of vasculature and fibrous tissue, and the outer layer of the SS (SS-Outer) layer consisting of abundant fat tissue. By dissecting the gallbladder along the SS-Inner layer after exposure at a safe region, bile duct and/or vascular injury can be avoided, even in cholecystitis cases.

- Two method in cholecystectomy:

duct first method:

the cystic duct and artery are first dissected and divided

fundus first method:

in which dissection is started from fundus and gradually proceed toward cystic duct

Technique

In general, open cholecystectomy can be performed by either of two different approaches:

- Retrograde
- Anterograde

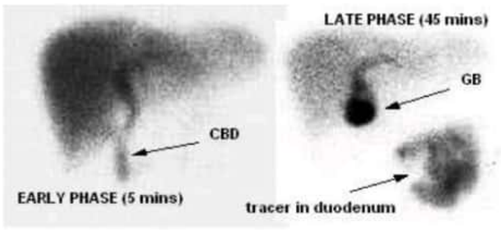
Radiology

Acute Cholecystitis

IOC: USG – Distended GB

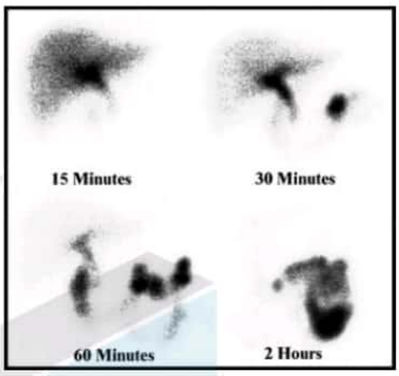
Murphy's Sign: Pressing the USG probe will elicit tenderness in the patient.

HIDA SCAN



Normal patient

Liver → Gall Bladder → Intestine



Non Visualization of Gall Bladder:

Seen in **Acute Cholecystitis**

HIDA Scan:

- Hepatobiliary imino-diacetic acid (HIDA)
- Evaluate the physiologic secretion of bile GB function
- Inject HIDA- process in liver-excrete in bile
- Failure to fill the gall bladder 2 hours: obstruction of the cystic duct
- Normal Study: <60 mins visualize GB
- Delayed visualisation- Chronic Cholecystitis.

Biliary Dyskinesia

- CCK-stimulated HIDA scan : EF < 1/3rd at 20mins diagnostic of dyskinesia,
- which should be managed with cholecystectomy

Contraindication of Lap Chole is GB Carcinoma where Open Chole is done .

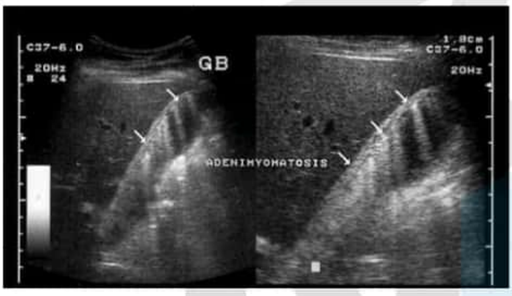
Indications of prophylactic cholecystectomy

- Sickle cell disease
- TPN
- Chronic immunosuppression
- Acalculous cholecystitis
- Gallstone pancreatitis
- Gall bladder polyp > 10mm
- Porcelain gall bladder
- No immediate access to health care facility

Gall Stones

i. Black Pigment Stones: Hemolytic stones (Radio-opaque) made up of Calcium phosphate and calcium bicarbonate.
ii. Brown Pigment Stones: because of Infection (Radio-opaque stones) made up of Calcium salts like palmate, stearate, and bilirubin complex.
iii. Mixed – MOST COMMON

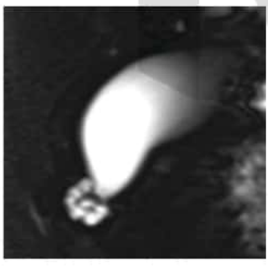
PORCELAIN (Calcified) Gall Bladder seen on XRAY



"Comet Tail Sign"

Seen on USG in ADENOMYOMATOSIS of Gall Bladder due to the Rokitansky sinuses

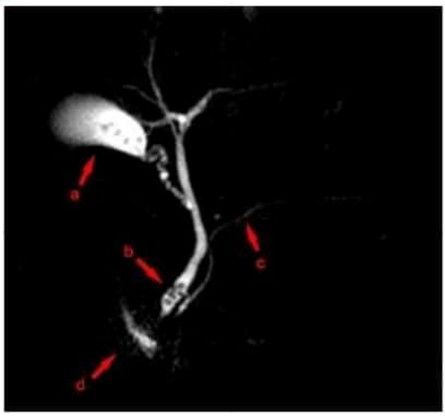
Similar disease in uterus is Adenomyosis where they show VENETIAN Blinds sign.



"Rosary Bead Sign" seen on MRCP in Adenomyomatosis of Gall Bladder

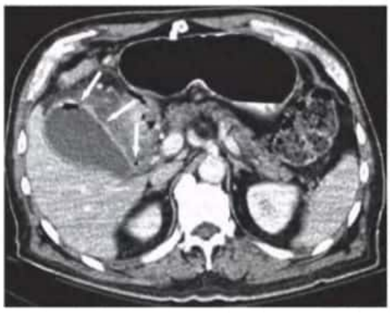
MRCP

- For CBD pathologies
- Non invasive technique
- Heavy T2 weighted MRI



MRCP image showing stones in the distal common bile duct: (a) Gallbladder with stones, (b) Stones in bile duct, (c) Pancreatic duct, (d) Duodenum.

Diverticulosis of Gall Bladder



Empyema of the Gall Bladder

CBD Stones

i. Asymptomatic (silent)
ii. Symptomatic- CHARCOT'S TRIAD
   a. Biliary Colic (Pain)
   b. Obstructive Jaundice
   c. Fever
iii. If left untreated, can lead to REYNOLD's Pentad:
Charchot's Triad + Hypotention + Altered Mental Status

ERCP

- Indication of Pre-Op ERCP (before cholecystectomy)
1. Cholangitis
2. Biliary pancreatitis
3. Planned for CBD exploration
4. Multiple Co-Morbidity

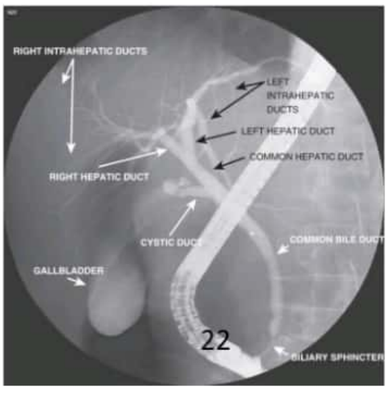
Investigation:

- On USG, a CBD >7-8 mm diameter indicates distention of the CBD
- ERCP- diagnostic and therapeutic
  - M/C complication a/w ERCP → Acute Pancreatitis
- MRCP- Sensitivity >90%, Specificity 100%

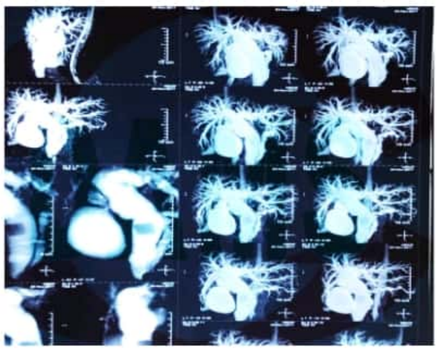
Causes of failure of ERCP

1. Large stone > 1cm
2. Multiple stone > 8
3. Altered gastric duodenal anatomy
4. Impacted stone
5. Duodenal diverticula

Side View Endoscopy



Side view endoscope is preferred for ERCP over a front view endoscope



ERCP with Stenting

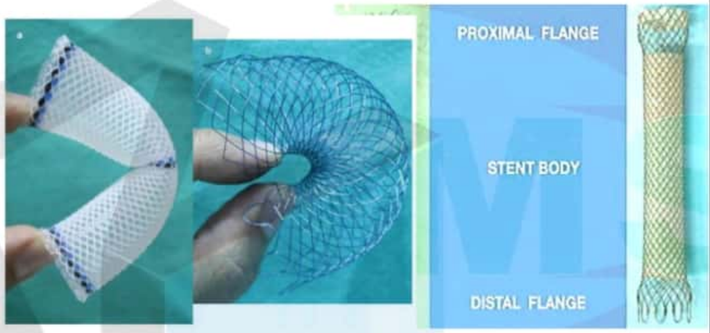
(A stent dilates, a shunt cross-connects)

Endo GI Stent

TYPES

1. SEMS uncovered
2. SEMS partially covered
3. SEMS completely covered
4. SEPS

Metallic is always preferred.

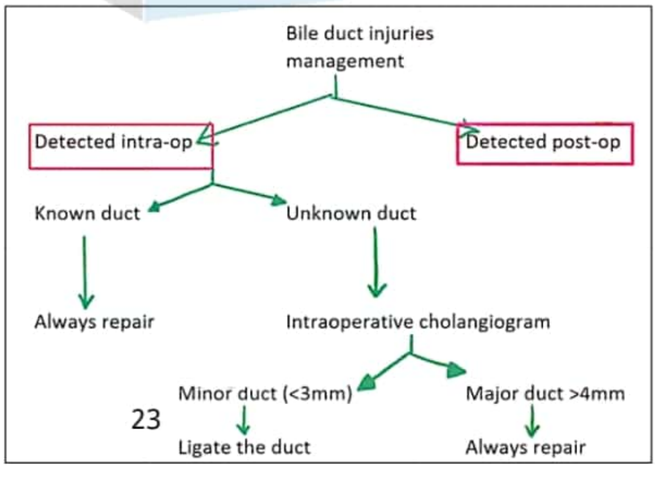


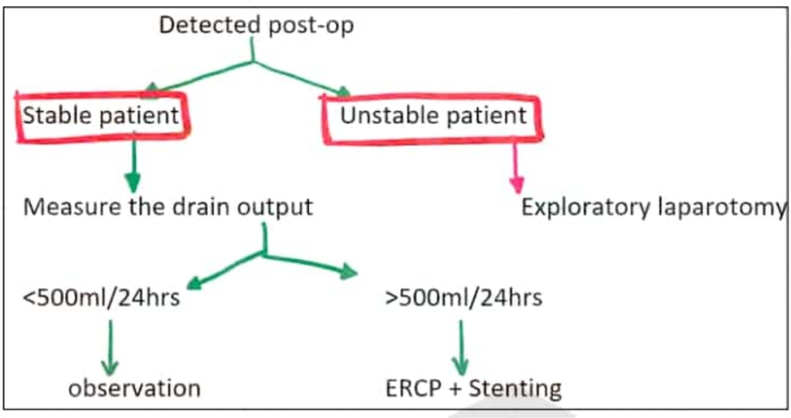
Bile Duct Injuries

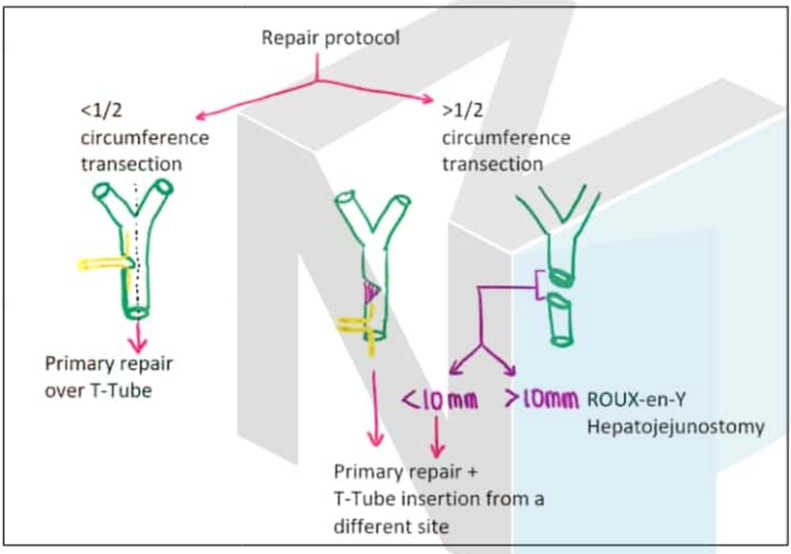
Case: A surgeon encounters difficulty during an elective laparoscopic cholecystectomy in a healthy 25-year-old woman and converts to an open procedure. The 4-mm common hepatic duct has been transected 1cm below the bifurcation. Which of the following procedures is the most appropriate?

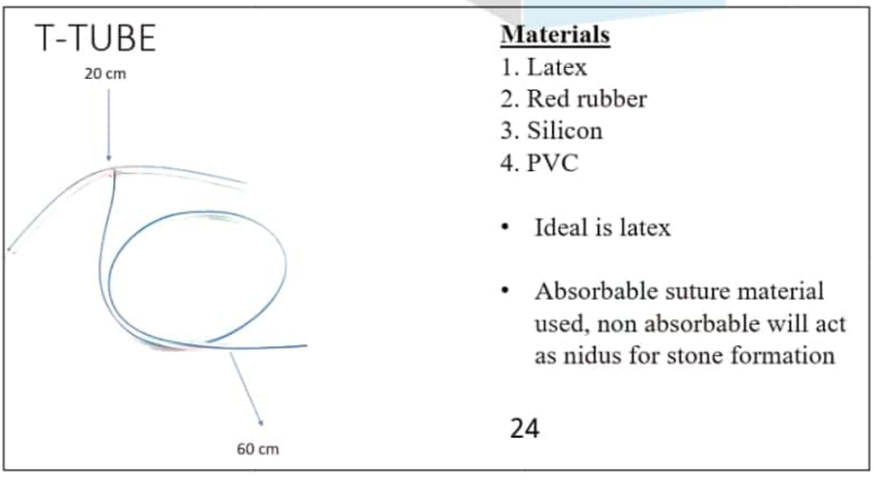
a. Duct-to-duct repair over a T tube
b. Duct to Duct repair without a stent
c. **Roux-en-Y-hepaticojejunostomy**
d. Hepaticoduodenostomy

M/C cause for Bile duct injures is **Poor Visual Perception**







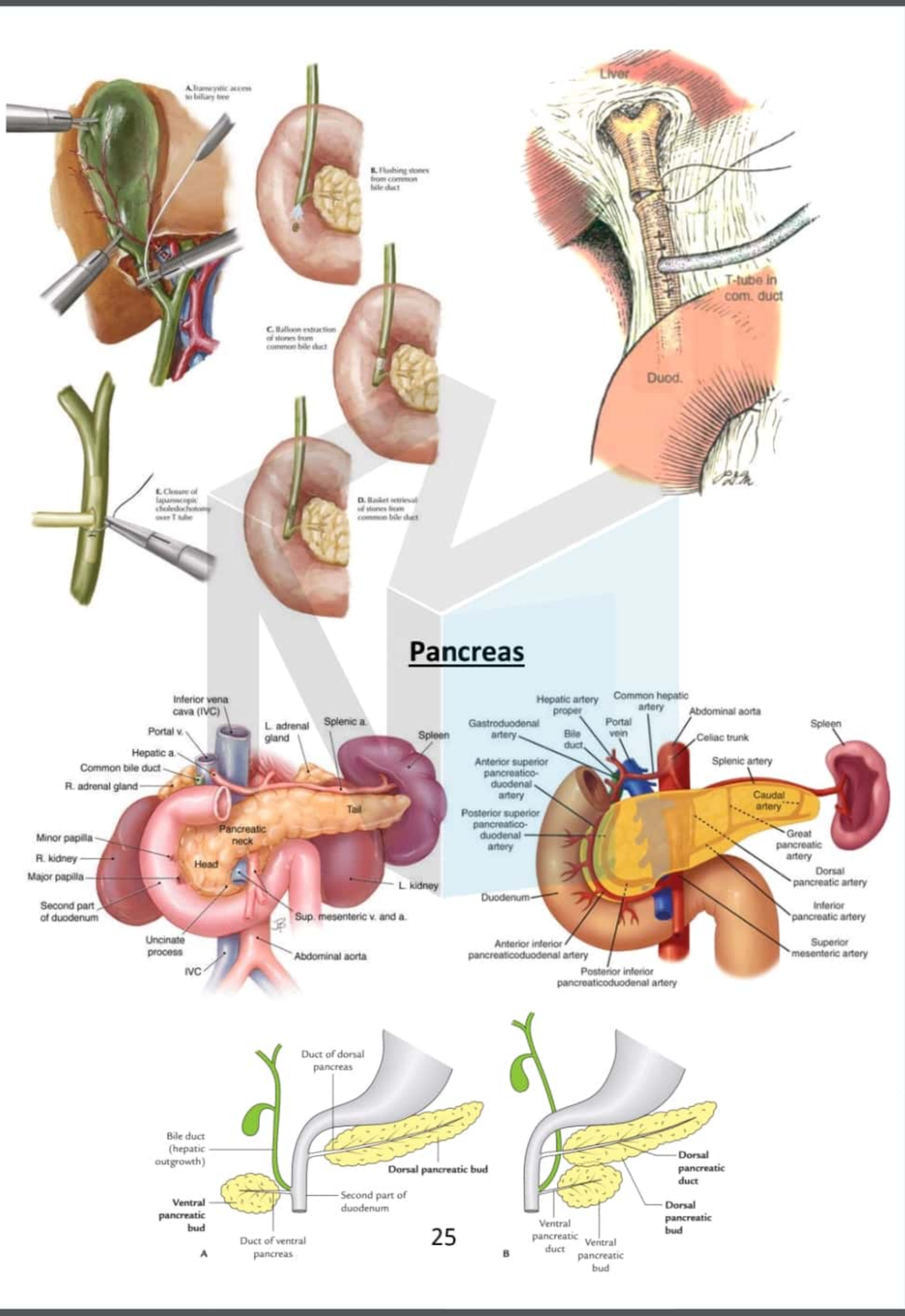


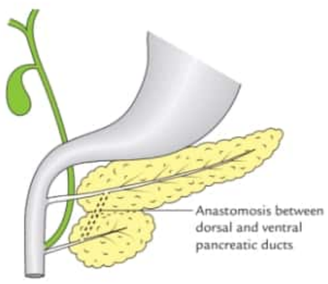
Materials

1. Latex
2. Red rubber
3. Silicon
4. PVC

- Ideal is latex
- Absorbable suture material used, non absorbable will act as nidus for stone formation

24


A. Transcystic access to biliary tree
B. Flushing stones from common bile duct
C. Balloon extraction of stones from common bile duct
D. Basket retrieval of stones from common bile duct
E. Closure of laparoscopic choledochotomy over T tube
Liver
T-tube in com. duct
Duod.
Pancreas
Inferior vena cava (IVC)
Portal v.
Hepatic a.
Common bile duct
R. adrenal gland
L. adrenal gland
Splenic a.
Spleen
Tail
Pancreatic neck
Head
Minor papilla
R. kidney
Major papilla
Second part of duodenum
Uncinate process
IVC
L. kidney
Sup. mesenteric v. and a.
Abdominal aorta
Hepatic artery proper
Common hepatic artery
Abdominal aorta
Gastroduodenal artery
Bile duct
Portal vein
Celiac trunk
Spleen
Anterior superior pancreatico-duodenal artery
Splenic artery
Posterior superior pancreatico-duodenal artery
Caudal artery
Great pancreatic artery
Dorsal pancreatic artery
Duodenum
Inferior pancreatic artery
Anterior inferior pancreaticoduodenal artery
Superior mesenteric artery
Posterior inferior pancreaticoduodenal artery
Duct of dorsal pancreas
Bile duct (hepatic outgrowth)
Dorsal pancreatic bud
Ventral pancreatic bud
Second part of duodenum
Duct of ventral pancreas
A
Dorsal pancreatic duct
Dorsal pancreatic bud
Ventral pancreatic duct
Ventral pancreatic bud
B



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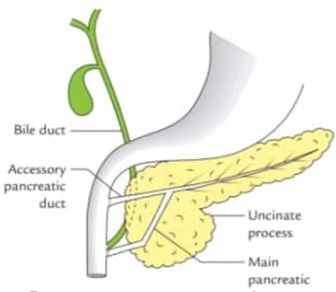
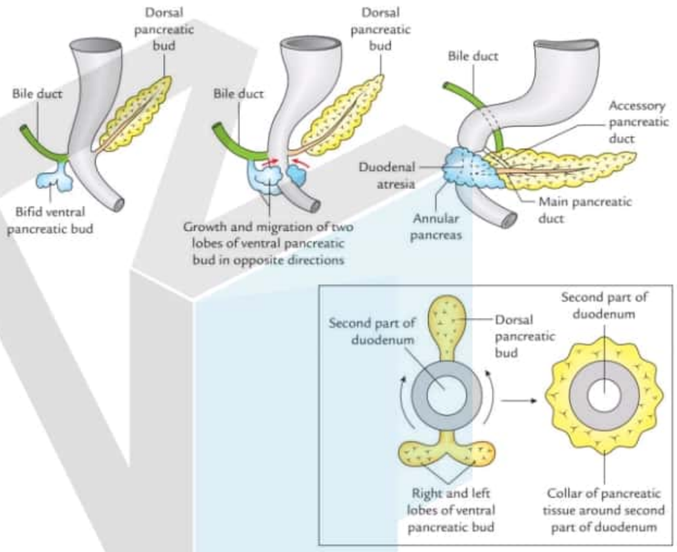


D

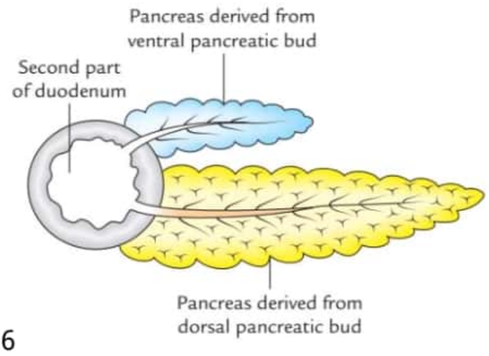
Annular Pancreas

In this condition, the pancreatic tissue completely surrounds second part of the duodenum causing its obstruction. This anomaly is produced as follows: The bifid ventral pancreatic bud fails to fuse to form a single mass. The two lobes (right and left) of the ventral pancreatic bud grow and migrate in opposite directions around the second part of the duodenum and form a collar of pancreatic tissue before it fuses with dorsal pancreatic bud. Thus, duodenum gets completely surrounded by the pancreatic tissue that may cause *duodenal obstruction.*


Dorsal pancreatic bud
Bile duct
Bifid ventral pancreatic bud
Dorsal pancreatic bud
Bile duct
Growth and migration of two lobes of ventral pancreatic bud in opposite directions
Bile duct
Accessory pancreatic duct
Duodenal atresia
Main pancreatic duct
Annular pancreas
Second part of duodenum
Dorsal pancreatic bud
Second part of duodenum
Right and left lobes of ventral pancreatic bud
Collar of pancreatic tissue around second part of duodenum

Pancreatic Divisum

It occurs when the dorsal and ventral pancreatic buds fail to fuse with each other. As a result, the two parts of pancreas derived from two buds remain separate from each other.



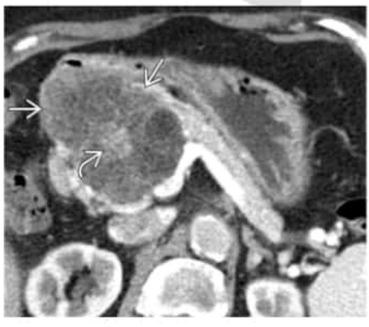
Benign Lesions

Defining Characteristics of Pseudocysts and Pancreatic Cystic Neoplasms

CHARACTERISTICS	PSEUDOCYST	SCN	MCN	IPMN
Epidemiology				
Gender	F = M	F ≫ M (4:1)	F ≫> M (10:1)	F = M
Age (years)	40-60	60-70	50-60	60-70
Imaging Findings				
Location	Evenly distributed	Evenly distributed	Head ≪ body/tail	Head > diffuse > body/tail
Appearance	Round, thick-walled large cyst; gland atrophy ± calcification	Multiple small cysts separated by internal septations with central starburst calcifications	Thick-walled, septated macrocyst with smooth contour; ± solid component, eggshell calcifications	Poorly demarcated, lobulated, polycystic mass with dilation of main or branch ducts
Communication with ducts	Yes	No	Very rare	Yes
Cyst Fluid Analysis				
Cytology	Inflammatory cells	Scant glycogen-rich cells, with positive periodic acid–Schiff stain	Sheets and clusters of columnar, mucin-containing cells	Tall, columnar, mucin-containing cells
Mucin stain	Negative	Negative	Positive	Positive
Amylase	Very high	Low	Low	High
CEA	Low	Low	High	High

- Pseudocyst: a minimum of 6 mm wall thickness and 6cm of internal diameter (Rule of 6)
- Amylase is high only in main duct IPMN. It is relatively less in branch duct IPMN.
- Management:
  - Pseudocyst- Cystojejunostomy or cystogastrostomy
  - SCN- Enucleation
  - MCN- Distal pancreatectomy
  - IPMN- Local resection or Distal pancreatectomy

Radiology



Pancreatic Serous Cystadenoma

Multicystic lesion

Central Stellate scar (Sunburst calcific scar)

IPMN: Duct is dilated
(Not dilated in cystadenoma)

Pseudocyst of Pancreas

Psuedocyst term used only after 4 weeks of Acute Edematous Pancreatitis.
For acute Necrotising pancreatitits, term used is 'walled off necrosis'

Chronic Pancreatitis

- TIGAR- O-Classification
- Other important ones are: Ranson's criteria, APACHE score
- MRCP with Secretin shows- **Chain of Lakes Appearance** →→
- **Sausage shape pancreas on CT** is seen in AUTOIMMINE pancreatitis

Chronic Pancreatitis and Pancreatic Exocrine Insufficiency: Tigar-O Classification System

- **Toxic-metabolic**
  - Alcoholic
  - Tobacco smoking
  - Hypercalcemia
  - Hyperlipidemia
  - Chronic renal failure
  - Medications—phenacetin abuse
  - Toxins—organotin compounds (e.g., DBTC)
- **Idiopathic**
  - Early onset
  - Late onset
  - Tropical
- **Genetic**
  - Hereditary pancreatitis
  - Cationic trypsinogen
  - PRSS$_1$
  - PRSS$_2$
  - CFTR mutations
  - SPINK1 mutations
- **Autoimmune**
  - Isolated autoimmune chronic pancreatitis
  - Autoimmune chronic pancreatitis associated with Sjögren's syndrome
  - Inflammatory bowel disease
  - Primary biliary cirrhosis
- **Recurrent and Severe Acute Pancreatitis**
  - Postnecrotic (severe acute pancreatitis)
  - Recurrent acute pancreatitis
  - Vascular diseases/ischemia
  - Postirradiation
- **Obstructive**
  - Pancreas divisum
  - Sphincter of Oddi disorders (controversial)
  - Duct obstruction (e.g., tumor)
  - Preampullary duodenal wall cysts
  - Posttraumatic pancreatic duct scars

Abbreviations: DBTC, dibutylin dichloride; TIGAR-O, toxic-metabolic, idiopathic, genetic, autoimmune, recurrent and severe acute pancreatitis, obstructive.

Genetic also includes Chymotrypsin-c gene

Radiology

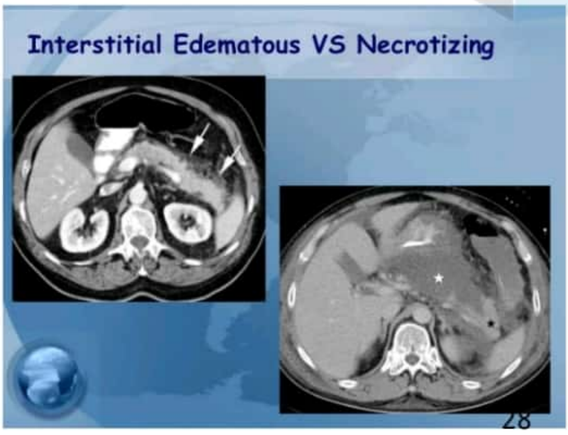
Colon Cutoff sign

Transverse colon appears to abruptly end due to spasm of the descending colon.

Seen in Plain XRAY of Acute Pancreatitis

Sentinel Loop Sign

Dilated one or two bowel loops indicating inflammation due to ileus.
Seen on plain XRAY in Acute pancreatitis.



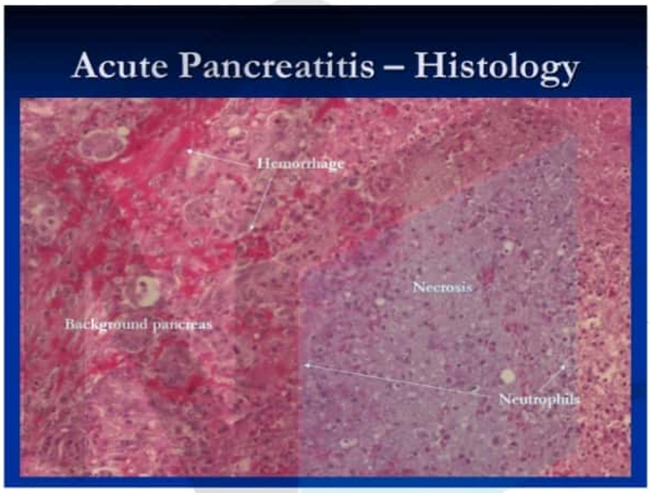
MODIFIED CT SEVERITY INDEX

PROGNOSTIC INDICATOR	POINTS
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic necrosis	
None	0
≤30%	2
>30%	4
Extrapancreatic complications (one or more of the following: pleural effusion, ascites, vascular complications, parenchymal complications or gastro-intestinal tract involvement)	2

Table 19.2 Inherited Predisposition to Pancreatitis

Gene (Chromosome Location)	Protein Product	Function
CFTR (7q31)	Cystic fibrosis transmembrane conductance regulator	Epithelial anion channel. Loss-of-function mutations alter fluid pressure and limit bicarbonate secretion, leading to inspissation of secreted fluids and duct obstruction
PRSS1 (7q34)	Serine protease 1 (trypsinogen 1)	Cationic trypsin. Gain-of-function mutations prevent self-inactivation of trypsin
SPINK1 (5q32)	Serine peptidase inhibitor, Kazal type 1	Inhibitor of trypsin. Mutations cause loss-of-function, increasing trypsin activity
CASR (3q13)	Calcium-sensing receptor	Membrane-bound receptor that senses extracellular calcium levels and controls luminal calcium levels. Mutations may alter calcium concentrations and activate trypsin
CTRC (1p36)	Chymotrypsin C (caldecrin)	Degrades trypsin, protects the pancreas from trypsin-related injury
CPA1 (7q32)	Carboxypeptidase A1	Exopeptidase involved in regulating zymogen activation

Autoimmune pancreatitis is associated with Ig-G4 related diseases



Indications for surgery in chronic pancreatitis:

a. Intractable pain
b. Symptomatic local complications
c. Unsuccessful endoscopic management
d. Suspicion of malignancy ***

Types of surgeries:

i. Pure drainage:
   a. Puestow procedure
   b. LPJ- (Latero-lateral pancreatico-jejunostomy)
   c. Parington-Rochelle procedure
ii. Duodenal preserving pancreatic head resection (DPPHR)
   a. Beger's: Head Resection
   b. Bern's: Head Resection
   c. Frey's: Head Coring

Pancreatic Adenocarcinoma

- M/C site: Head
- Age: 6th decade
- M/C type: Pancreatic ductal Adenocarcinoma
- Most characteristic feature: Waxing and Weaning of Jaundice (d/t hypertrophy and sloughing of the tumour)
- TROSSUE Syndrome: Migratory superficial thrombophlebitis seen in Ca Stomach, Ca pancreas and Thromboangitis obliterans (TAO)
- TROSSIER Sign: Enlarged Left supraclavicular group of lymph nodes (Virchow's Nodes) seen in Ca Stomach. (Left Axillary LN- Irish group of LN)
- TROSSUE Sign: Carpopedal spasm seen in HYPOCALCEMIA

The **double duct sign** refers to the presence of simultaneous dilatation of the common bile and pancreatic ducts. The two most common causes of the double duct sign are carcinoma of the head of the pancreas and ampullary tumors (e.g. carcinoma of the ampulla of Vater).

Adenoma-Carcinoma Sequence (Also seen in Colon Ca)

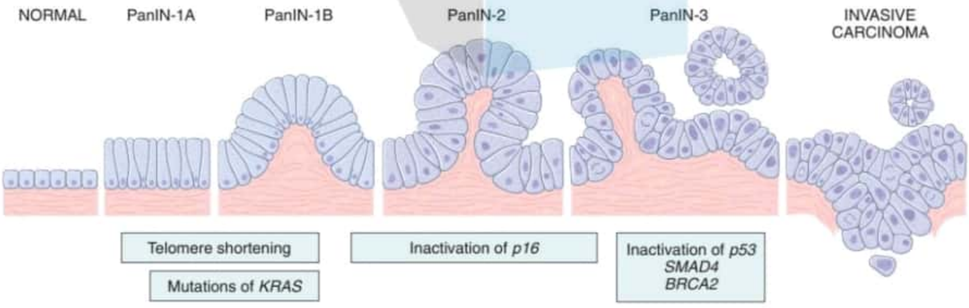
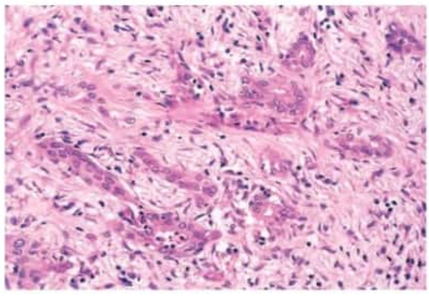


Fig. 17.8 Progression model for the development of pancreatic cancer. It is postulated that telomere shortening and mutations of the *KRAS* oncogene occur at early stages, inactivation of the p16 tumor suppressor gene occurs at intermediate stages, and inactivation of the *TP53*, *SMAD4*, and *BRCA2* tumor suppressor genes occurs at late stages. Note that while there is a general temporal sequence of changes, the accumulation of multiple mutations is more important than their occurrence in a specific order. PanIN, Pancreatic intraepithelial neoplasm. The numbers following the labels on the top refer to stages in the development of PanINs. *(Modified from Maitra A, Hruban RH: Pancreatic cancer,* Annu Rev Pathol Mech Dis *3:157, 2008.)*

M/C Mutation: p16; 2nd M/C mutation: KRAS

Table 19.4 Inherited Predisposition to Pancreatic Cancer

Disorder	Gene	Increased Risk of Pancreatic Cancer (Fold)	Risk of Pancreatic Cancer by Age 70 (%)
Peutz-Jeghers syndrome	*STK11*	130	30–60
Hereditary pancreatitis	*PRSS1, SPINK1*	50–80	25–40
Familial atypical multiple-mole melanoma syndrome	*CDKN2A*	20–35	10–17
Strong family history (3 or more relatives with pancreatic cancer)	Unknown	14–32	8–16
Hereditary breast and ovarian cancer	Multiple, including *BRCA1, BRCA2, PALB2, ATM*	4–10	5
Hereditary non-polyposis colorectal cancer	Multiple, including *MLH1, MSH2, PMS2*	8–10	4



HPE: Poorly formed glands composed of malignant epithelial cells are dispersed within densely fibrotic stroma; some inflammatory cells are also present.

Borderline Resectable Pancreatic Cancer Criteria (NCCN Guidelines)

Borderline Resectable[2]	Pancreatic head/uncinate process: • Solid tumor contact with CHA without extension to celiac axis or hepatic artery bifurcation allowing for safe and complete resection and reconstruction. • Solid tumor contact with the SMA of ≤180° • Solid tumor contact with variant arterial anatomy (ex: accessory right hepatic artery, replaced right hepatic artery, replaced CHA, and the origin of replaced or accessory artery) and the presence and degree of tumor contact should be should be noted if present as it may affect surgical planning. Pancreatic body/tail: • Solid tumor contact with the CA of ≤180° • Solid tumor contact with the CA of >180° without involvement of the aorta and with intact and uninvolved gastroduodenal artery thereby permitting a modified Appleby procedure [some members prefer this criteria to be in the unresectable category].	• Solid tumor contact with the SMV or PV of >180°, contact of ≤180° with contour irregularity of the vein or thrombosis of the vein but with suitable vessel proximal and distal to the site of involvement allowing for safe and complete resection and vein reconstruction. • Solid tumor contact with the inferior vena cava (IVC).

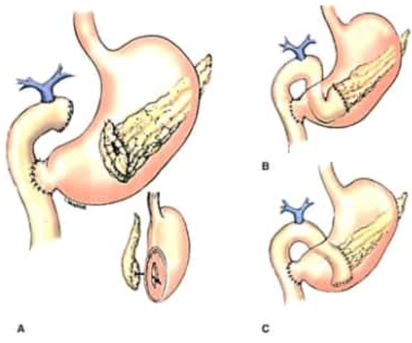
Abutment: Tumour growth encroaches a vessel, the fat place is disrupted but the vessel truly is not involved.

Infiltration: Tumour growth encroaches a vessel, and the vessel is involved.

If borderline resectable: we do Pancreato-duodenectomy (Whipple's technique)

Whipples Surgery

- Most commonly the reconstruction first involves the pancreas, followed by the bile duct and then the duodenum or stomach.
- The most common reconstruction involves the end of the divided jejunum brought up in a retrocolic position with creation of a pancreaticojejunostomy, followed by hepaticojejunostomy and then a duodenojejunostomy
- The pancreatic reconnection is the most problematic anastomosis of the three and responsible for much of the morbidity associated with the procedure

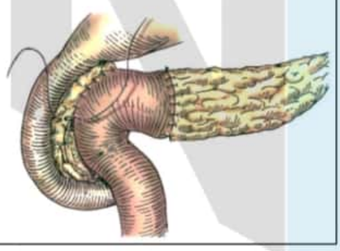


Whipple's Surgery: Tri-anastomosis: Pancreato-Jejunostomy, Hepato-Jejunostomy, and Duodenojejunostomy (if pylorus preserving) or Gastrojejunostomy (if non-pylorus preserving).

- 1st Anastomosis: Pancreato-Jejunostomy
- M/C complication: Anastomotic leak
- M/C cause of post-operative death: Anastomotic leak

Surgical Techniques		Indications and Recommendations
Pure Drainage Operations	Laterolateral pancreaticojejunostomy	Ductal dilation > 7 mm, without inflammatory mass
	Partington-Rochelle procedure	
	Puestow-procedure	Rare indications, replaced by other procedures
DPPHR Techniques	Beger	Procedures of choice if inflammatory mass in the head of the pancreas
	Bern	Bern technically less difficult than Beger but equal long-term outcome
	frey	Patients with ductal obstruction in the head and tail and a smaller inflammatory mass in the head

Begers



Frey's